Because epidemiology is entangled in our society we must take hold of this locus and be responsible for our research in its social context, a stance clearly in conflict with the writings of Rothman, who maintains that our focus should be on causation and that as a discipline we should avoid political or policy debates.

Epidemiology focuses on the distribution of ill-health as well as the social determinants of disease; consequently, it is not purely an observational discipline, but participatory as well. Research on passive smoking exemplifies the political rather than the purely scientific audience for research findings.

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APPENDIX 18
Percentage Distribution of Measured Tar Content[1] of Cigarette Brand Currently Smoked at Enrollment in CPS-I and CPS-II

Sex	Number[2] of Subjects	Percentage by Tar Content (mg)						
		<6.0 mg	6.0-11.9 mg	12.0-16.9 mg	17.0-20.9 mg	21.0-25.7 mg	25.8-35.7 mg	
Men								
CPS-I	128,427	—	0.4	12.4	29.2	17.5	40.6	
CPS-II	91,209	19.5	23.5	38.8	11.1	7.1	—	
Women								
CPS-I	135,604	—	0.7	25.3	34.9	15.7	23.3	
CPS-II	123,442	18.9	31.7	38.6	7.5	3.3	—	

[1] Tar content based on Garfinkel, 1979, and Federal Trade Commission, 1983.
[2] Excludes cigarette smokers who did not specify the brand of cigarettes currently smoked. Total number of smokers shown in Table 4.
Key: CPS = Cancer Prevention Study.

APPENDIX 19
Age-Specific Deaths and Death Rates From Lung Cancer as Underlying Cause Among Lifelong Never-Smokers and Current Cigarette Smokers: Men, CPS-I and CPS-II

	CPS-I				CPS-II			
	Never-Smokers		Current Cigarette Smokers		Never-Smokers		Current Cigarette Smokers	
Age	Deaths	Rate[1]	Deaths	Rate[1]	Deaths	Rate[1]	Deaths	Rate[1]
35-39	0	—	3	8.1	1	4.6	1	5.0
40-44	1	4.0	16	28.1	0	—	4	18.7
45-49	4	8.0	66	45.6	4	8.0	26	41.4
50-54	6	5.7	162	75.0	7	5.5	136	115.3
55-59	13	13.6	217	131.5	7	5.3	260	206.1
60-64	16	21.0	229	231.2	14	11.6	381	361.1
65-69	14	23.1	183	341.1	22	21.5	400	581.6
70-74	13	29.7	97	403.4	25	34.9	343	909.0
75-79	6	31.0	53	612.7	21	52.0	170	1,118.3
80-84	6	67.5	6	334.9	16	89.2	51	1,227.7
85+	2	35.3	1	178.6	7	66.6	9	919.0
Total	85		1,035		124		1,781	

[1] Death rate per 100,000 person-years.
Key: CPS = Cancer Prevention Study.

Table 14
Percentage of the total rate difference for all causes between smokers and never-smokers contributed by various diseases[a]

Disease	Percent in Men		Percent in Women	
	CPS-I	CPS-II	CPS-I	CPS-II
Lung Cancer	14.7	28.1	6.8	28.0
Coronary Heart Disease	41.7	21.8	46.6	19.0
Chronic Obstructive Pulmonary Disease	5.6	8.2	6.1	11.2
Stroke	4.7	4.3	10.3	6.7
Other Smoking-Related Cancers	5.6	7.4	8.4	6.4
Other Conditions	27.7	30.2	21.8	28.7

[a] *Based on the cause-specific rate difference divided by the all-cause rate difference in Tables 5 and 8 through 12.*

Key: CPS = Cancer Prevention Study.

Caution is urged in interpreting the comparison of lung cancer death rates at "equivalent" levels of self-reported smoking. Information on the number of cigarettes smoked at enrollment may not mirror the lifelong patterns of smoking that cause lung cancer. Cigarette consumption during adolescence and early adulthood was probably heavier among smokers in CPS-II than in CPS-I for several reasons. First, manufactured cigarettes were more available in the 1940's and 1950's than in the 1920's and 1930's (U.S. Department of Health and Human Services, 1989). CPS-II smokers born in the late 1920's typically began smoking after World War II when cigarettes were plentiful and there were few prohibitions against smoking. Second, birth cohort analyses of the U.S. general population show that the peak prevalence of smoking among white men increased with each successive birth cohort from 1900 to 1929 and decreased thereafter (Burns, 1994). Similarly, age-specific death rates from lung cancer death rates have decreased among U.S. men born after 1930 (Devesa et al., 1989; Gilliland and Samet, 1994). Thus, the large increase in death rates from CPS-I to CPS-II probably reflects unmeasured heavier smoking in CPS-II during the 1940's and 1950's as well as the measured increase in daily consumption and duration of smoking.

Other factors that could influence the intensity of cigarette smoking are that CPS-II smokers may include more addicted "hard core" smokers who cannot quit despite health and social concerns. Partly to compensate for the lower tar and nicotine content of modern cigarettes (U.S. Department of Health and Human Services, 1989), CPS-II smokers may inhale more deeply, take more puffs per cigarette, or retain the smoke longer in their lungs than did smokers in the past (Benowitz et al., 1983 and 1986; Herning et al., 1981; Russell et al., 1980; U.S. Department of Health and Human Services, 1988). Strong social prohibitions against smoking may have caused CPS-II smokers to underreport usage or to reduce their consumption in an effort to quit.

Smokers in the 1980's also may have been more vulnerable to the carcinogens in tobacco smoke because of lower dietary intake of fresh fruits and vegetables (Subar et al., 1990; Willett, 1990). Finally, the large decrease in cardiovascular mortality from CPS-I to CPS-II could contribute somewhat to the increasing lung cancer death rates, although most potential confounding resulting from competing causes was eliminated by stratifying person-years at risk into 5-year age intervals.

Despite the many uncertainties that constrain the ability to compare the intrinsic carcinogenicity of cigarettes from these two eras, the net effect of all changes in the cigarette and the smoking of cigarettes has been a large increase rather than decrease in lung cancer mortality in smokers. Although low-tar, filter-tip cigarettes have been shown to slightly reduce lung cancer risk compared with nonfiltered cigarettes in several epidemiologic studies (Hammond, 1980; Hammond et al., 1976; Lubin et al., 1984a and 1984b; Vutuc and Kunze, 1982; Wynder et al., 1970), the potential benefits of these products are clearly overwhelmed by the more potent adverse changes in smoking behavior and perhaps by other unidentified factors. The evaluation of cigarettes has not protected smokers from fatal lung cancer.

The falling death rates from CHD and stroke seen in this study reflect major nationwide declines that began for CHD in the mid-1960's and for stroke in the 1940's or earlier (Cooper et al., 1978; Higgins and Thom, 1989; Moriyama et al., 1971; Ragland et al., 1988; Russell et al., 1980). Data suggest that much of the decline results from factors other than smoking cessation because mortality decreased among both current smokers and lifelong never-smokers, groups largely unaffected by smoking cessation. Much of the nationwide decline in CHD mortality probably reflects reduced mortality resulting from therapeutic advances. We measured mortality rather than incidence and could not distinguish between changes in incidence because of diet, exercise, antihypertensive or antithrombotic therapy, control of lipids, or improvement in survival because of medical care. Most of these factors, as well as smoking cessation, are thought to play some role in the nationwide CHD decline (Cooper et al., 1978; Higgins and Thom, 1989; Moriyama et al., 1971; Ragland et al., 1988; Russell et al., 1980), although their relative importance is unknown.

Because CPS-I and CPS-II include mostly white middle-class Americans (Garfinkel, 1985; Stellman and Garfinkel, 1986), it cannot be concluded that the trend of falling CHD mortality will affect all segments of the U.S. population equally. For the poor and minorities in particular, more limited access to medical treatment and prevention may result in a slower decline in CHD mortality (Cooper et al., 1978) and proportionately greater CHD mortality as a cause of excess death in smokers. Because the poor are increasingly overrepresented among the 46 million Americans who smoke, the authors' data may underestimate the excess in CHD mortality among smokers in the general population.